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UNITED STATES DISTRICT COURT
DISTRICT OF MASSACHUSETTS

- x

JOANNE KOTLER, Individually and as Administratrix of the Estate of GEORGE P. KOTLER,
Plaintiff,

v.

Civil Action
No. 86-0810-S

THE AMERICAN TOBACCO COMPANY, PHILIP MORRIS, INC. and LIGGETT GROUP, INC.,
Defendants.

- x

BEFORE: Honorable Walter Jay Skinner

Held At:

John W. McCormack
Post Office and Courthouse
Boston, Massachusetts
Friday, March 2, 1990
9:05 a.m.

DORIS M. JONES & ASSOCIATES, INC.
Professional Shorthand Reporters
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APPEARANCES:

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I N D E X

WITNESSES:

EXHIBITS:	DESCRIPTION	FOR ID.	IN EVID.

P R O C E E D I N G S.

THE COURT: Counsel wanted to see me?

(Side bar conference.)

MR. BEZANSON: Good morning, your Honor.

The Defendant would like to hand up a memorandum of law pointing out that Sharp did not reek a change in Massachusetts law, but as is shown by a Supreme Judicial Court opinion cited a week after Sharp and two Massachusetts Court of Appeals cases decided since Sharp, the but for standard for approximate cause remains the law in Massachusetts.

THE COURT: We're in our usual state of shambles again.

MR. BEZANSON: No, they worked it all out on the but for.

THE COURT: I will -- have you given copies?

MS. LUMSDEN: I just got a copy, your Honor.

THE COURT: At this stage I'm sticking with my ruling. I assume that you'll renew your various motions and so forth at the end of all

young guy on the right or the student.

MR. LANE: I think he's the young fellow.

THE COURT: Ex-college student.

Is that all right with you?

MR. LANE: Fine with me.

THE COURT: Let's bring the jurors down, and we'll start with whoever we've got.

(End of side bar discussion.)

(Jury present at 9:25 o'clock a.m.)

THE COURT: Good morning, ladies and gentlemen.

We're going to press forward even in the absence of Mr. O'Connell. If he shows up I would excuse him. You may be seated. We're that close to the end of the trial that we think we can take the chance that we'll all survive another few days. There's just enough of you to make a jury. Then we'll press forward with the Defendants' case.

The witness?

MR. SHEFFLER: Your Honor, the American Tobacco Company would like to call Doctor McHugh to the stand.

THE CLERK: Please raise your right

hand.

DOCTOR PETER MC HUGH,

having been first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION

BY MR. SHEFFLER:

Q. Doctor, could you state your name for the record, please?

A. Peter McHugh.

Q. And what is your occupation?

A. I'm a surgical pathologist and a medical researcher.

Q. Doctor, where do you perform your medical research?

A. I currently work at Jefferson Medical College in Philadelphia, Pennsylvania.

Q. What's your area of medical research, Doctor?

A. I'm interested in tumor stem cells.

Q. What does tumor stem cells have to do with cancer?

A. Well, cancers are composed of many, many cells, and they all begin with one cell which

proliferates, and that one cell that they begin with is called a tumor stem cell.

Q. Doctor, if you could, I know you have a cold, if you could keep your voice up and speak into the microphone.

How long have you been doing this research on tumor stem cells?

A. Approximately ten years.

Q. And you mentioned also, Doctor, that you're a medical doctor.

Are you licensed to practice medicine?

A. Yes, I am.

Q. What area of medicine do you specialize in?

A. I specialize in pathology.

Q. Are you certified in pathology, Doctor?

A. Yes, I am. I'm board certified.

Q. When did that occur?

A. Nineteen eighty-two.

Q. Doctor, would you briefly describe for the jury your formal education beginning with college?

A. I attended undergraduate studies at Duke University, graduated with a degree in chemistry. I then went to Masonic School of Medicine in New York City, received my MD in 1977, and then did an

Q. Doctor, when you do research on molecular biology, did you say that involved genes, looking at genes and so?

A. Yes, it does. It's different than looking at tissues on the microscope, which is what I do at the hospital.

We have to use certain specialized techniques that have recently been developed to isolate the DNA, to cut it up in usable pieces, use an enzyme called restriction endonucleases. We have to do gel electrophoresis to separate these pieces. Then to identify the pieces we do specialized techniques called northern and southern hibernizations.

Q. Well, Doctor, if I may, is molecular biology related to cancer?

A. Absolutely. I think that the greatest strides in understanding cancer in the last five years have come by utilizing the techniques of molecular biology.

I think nowadays most people recognize that cancer is a genetic disease, and that is our best model to date. Certainly the Nobel Prize in medicine this year was given to Doctor Michael

Bishop out in California for his work in this area.

Q. By "genetic disease," what does that refer to, Doctor?

A. Essentially an alteration in the genes of the cell.

Q. Doctor, how did it come about that you were able to take a fellowship in molecular biology?

A. This particular fellowship was a project sponsored by the National Institutes of Health where one would submit a research proposal, that proposal would then be reviewed by a panel of scientists, given a priority score, and if the score was sufficient enough or high enough then they would fund it.

Q. Doctor, what was your research proposal and what research did you do at the Roche Institute?

A. The research that I did had to do with the effects of vitamin A on tumor stem cells.

Q. Did you publish papers as a result of your research, Doctor?

A. Multiple papers.

Q. You continue to do research in this area?

A. Yes. We continue to look at the effects of vitamin A on tumor stem cells, and we have a paper

coming out later this year in teratology on neuropress stem cells and the effects of vitamin A.

Q. Is the teratology a peer review journal, Doctor?

A. Yes, it is.

Q. Could you tell us approximately how many articles you've published in peer review journals, medical or scientific?

A. Currently we're probably about twenty.

Q. Doctor, are you a peer reviewer yourself for medical or scientific journals?

A. Yes, I do. I review for several journals. I review for laboratory investigation, I review for the journal called Differentiation, and also for Archives of Pathology.

Q. Are you a reviewer in any other peer review process?

A. I also review grants. I have in the past reviewed grants for the National Science Foundation in Washington, and also for the March of Dimes Foundation.

Q. Doctor, these grant proposals that you've reviewed, what would be the areas that they would concern?

A. They are in my area of expertise, which is tumor stem cells.

Q. Doctor, after your fellowship in molecular biology, where did you go?

A. I took a position as an associate -- excuse me, an assistant professor at Emery University in Atlanta, Georgia.

Q. What did you do there?

A. I did a combination of things. I did hospital pathology, I taught medical students, and I did my medical research.

Q. And the courses you taught medical students, Doctor, what did they deal with?

A. Essentially we taught pathology in general, pathology as related to cancer and systemic organ pathology.

Q. And your research, Doctor, at Emery?

A. Continued to be in the field of tumor stem cells.

Q. When did you leave Emery?

A. I left Emery at the end of '87 and took a position at Jefferson Medical College as an associate professor of pathology and cell biology.

Q. Doctor, are you continuing to practice

surgical pathology?

A. Yes, I am. Essentially my duties are the same.

Q. How was your research at Jefferson College funded?

A. It's funded in three ways. It gets departmental money, institutional money and government funded money.

Q. Your teaching practice at Thomas Jefferson, what subjects does it involve?

A. It also involves pathology, pathology related to cancer and pathology related to organ systems.

Q. In your surgical practice, Doctor, are you called upon to make diagnoses from cytology?

A. Yes, I am.

Q. Are you also called upon to make diagnoses from lung specimens?

A. Yes. We see all sorts of lung specimens; lung cytology, lung aspirates, lung biopsies, whole lungs from pneumonectomy specimens.

Q. Now, Doctor, at my request, have you reviewed the medical records, the x-rays and the pathology materials that relate to Mr. Kotler's

cancer?

A. Yes, I have.

Q. And based upon that review, do you have an opinion as to the medical cause of Mr. Kotler's death?

A. Yes, I do.

Q. What is that opinion, sir?

A. I believe that his cause of death was directly related to his lung cancer.

Q. Doctor, in your review of Mr. Kotler's medical records,, and the other materials that you had available to you, was there anything that you saw that would in your opinion place him at a higher risk for lung cancer than the average person?

A. My review of the records, and although I don't know everything about Mr. Kotler's history, the one thing that stood out in the medical records was his history of smoking cigarettes.

Q. And, Doctor, do you believe that Mr. Kotler's history of smoking cigarettes was a significant risk for his lung cancer?

A. Yes, I do.

Q. And what's the basis of your opinion that smoking is a risk for lung cancer?

A. I think there have been numerous statistical and epidemiologic studies that have correlated cigarette smoking as a risk factor for development of lung cancer.

Q. Doctor McHugh, there was testimony in this case by Mr. Kotler's treating physician, the person who diagnosed his cancer and cared for him during the final illness, Doctor Hilgenberg, and Doctor Hilgenberg testified that in his opinion it was more probable than not that Mr. Kotler would not have had lung cancer had he quit smoking in 1966.

Do you agree with that?

A. Yes, I do.

Q. And what's the basis for your agreement?

A. Well, I think that opinion is it can be supported by three lines of evidence; statistical evidence, evidence gained from experiments in cell biology, and evidence gained from experiments in molecular biology.

Q. Well, how does molecular biology support your opinion?

A. Well, I think I can best answer that question by drawing on the blackboard, if I could.

(Pause.)

BY MR. SHEFFLER:

Q. Doctor, I know you have a cold, but if you could please try to keep your voice up.

A. In the normal course of events, mature cell types in organ systems come from precurser cells. This represents a cell. It's going along its life cycle, and according to some pre-programmed message that is contained within the DNA, within the genes of the cell, it will eventually branch off and divide, forming a mature cell type, which then assumes the normal bodily functions. And then, of course, the mature cell types divide out and have to be replaced by the stem cell precurser.

Q. Mature cells don't divide?

A. Usually not.

Now, as these precurser cells are going along in the life cycle, they are exposed to numerous environmental factors which can cause, as we say, genetic hits, and they can get alterations in the genes.

Now, if you're unlucky enough to accumulate enough hits of the right type, a cell will eventually turn to cancer. Once it turns to cancer, it then will uncontrol and proliferate, form

a mat of many, many cells, and eventually kill the host.

However, we know from molecular biology studies that the cells have what are very efficient mechanisms to repair themselves, and we know that a cellular defect or a genetic defect is not forever, and these things will repair themselves if given enough time. And these systems have evolved because the cell is such a complex mechanism that it's very easy to slip in errors. When you divide, all the DNA has been replicated, and there's lots of little places where errors can slip in. For an organism to survive it has to have repair mechanisms going on at all times.

Q. Doctor, how many genes are in a DNA of one cell?

A. First of all, not all DNA is a gene, but we have approximately 100,000 genes contained within our nucleus, and at any one time approximately 10,000 of those genes are actively providing information for life of the cell.

Q. Doctor, this process you talked about before the line that it becomes cancerous, you mentioned that there's repair going on.

What did you mean by that?

A. Well, just what I said before, that the DNA gets repaired and returns to normal, so any of the defects before the single cell turns cancerous are reversible.

However, once you get enough hits, this is called exposure, and the hits are repaired, then cancer is irreversible, and then once the cancer cells start to divide, doubles, quadruples and whatnot.

Q. Doctor, have they observed repair of genetic alterations experimentally?

A. Yes, both spontaneous repair and repair caused deliberately in the laboratory.

Q. Are we through with this part?

A. Yes.

(Pause.)

BY MR. SHEFFLER:

Q. Doctor, you mentioned that cell biology supports your opinion that Mr. Kotler would have avoided his cancer had he stopped smoking in 1966.

Could you explain that for the jury?

A. Sure. Again I've made a couple posters to demonstrate this, and it has to do with the concept

of cell turnover.

(Pause.)

BY MR. SHEFFLER:

Q. Doctor, what is this that we're looking at here?

A. This is a picture that I've had blown up from my teaching collection that I use on medical students.

Q. Did you take this picture, Doctor?

A. Yes. This is a picture I've taken through my microscope in my office, and it represents lung tissue.

Okay. Now, what we see here are two parts to the lung. We see the air tubes of the bronchi of the lung, what is traditionally known as the central portion of the lung, and we also have here the periphery of the lung or the alveolar air sacks. And this line, lace-like pattern, is the alveolar septi.

Q. Doctor, would you point out the air tube or the bronchus again, please?

A. Okay. Think of this as a thin slice of lung. It's been cut this way, and this is the air tube, and that kind of air tube is coming out at

you, and trying to cross-sect it, looking down at it. It's not complete. This part is out of the picture (indicating).

Q. Would you explain what kind of cells those are?

A. Okay. The cells we see here are what are known as adult cell types. In the air tubes we have an epithelial line composed of multiple cell layers, multiple types of cells, tall columnar cells with the cilia which is the fuzz at the top, the clear cells are what is known as oppa (ph) cells, they tend to make secretions around the bronchi. We have smooth muscle, we have blood vessels to feed it. The red stuff is the red blood cells within the blood vessels. And then, of course, we have the peripheral part of the lung composed of the alveolar septi.

Q. Are those air sacks, Doctor?

A. Yes. These are what are known as air sacks.

Q. What happens in that area, Doctor?

A. Well, the oxygen or the air gets taken down the air tubes and gets distributed to the air sacks, and then we have oxygen exchanged between the red

cells and the spaces.

Q. What are the cells out there on that side?

A. These cells are different from the cells of the bronchi, and these are what we call type one, type two pneumocytes.

Q. Doctor, how does that explanation, how does that work to support your opinion that Mr. Kotler would have avoided his cancer if he had quit smoking in 1966?

A. Well, you have to remember that these adult cells all turned over. In the lung, the normal turnaround rate for lung epithelium is between fifteen and 30 days, approximately a month. And so the cell that you see here today in this picture is not the cell that's going to be there tomorrow. And if this cell happens to be injured, this injured cell is not going to be here tomorrow, it will either die off or perhaps be repaired. So there is cell turnover going on in the lung at all times.

Q. What replaces those cells, Doctor?

A. Well, okay, it gets a little bit more complicated than just this picture. Because this is such a complex organism, we have a lot of backup in maintaining our bodily functions, and we have what

are known as cell pools to maintain our normal mature cell types, and we have levels of redundancy in the body that we have broken up into approximately three pools, we have the pleural stem cells, which will give to pre-committed stem cells, which will then give committed stem cells, which then give mature cells.

What happens in the normal course of events is that a mature cell will be happily exchanging oxygen, and in 30 days it dries out, then it is replaced by a division that occurs in the committed stem cell pool.

Now, the turnover rate, of course, of the stem cell pool is approximately 50 divisions, and if these mature cells last about a month, then we have high activity once every month.

Q. Biototic activity, what is that?

A. Cell division.

Q. The committed stem cells you're talking about, those divide how many times?

A. Once or twice. They renew each other. But essentially this pool lasts four to five years.

Now, in this pool, if a committed stem cell pool is used up, it is then replaced by the

pool in the pre-committed stem cell. And, of course, we have all this level of redundancy to maintain enough cells to last our lifetime. And also we have more in case there's any type of catastrophic injury, any type of trauma or any type of, say, overwhelming infection. We have to be able to regenerate.

Q. Doctor, what cells of those types you have up there in your opinion would most likely give rise to a cancer?

A. Well, it's usually the committed stem cell pool that is most likely to accumulate the genetic defects that would cause cancer, because there are probably the most mycotically active, and in the phase of cell division you're more susceptible to genetic hits.

Q. Doctor, what is the life span of those cells that are susceptible to genetic hits?

A. About four to five years.

So if these cells get genetic hits, if they don't accumulate the proper type and number of hits to form a cancer, in four to five years they are going to be gone and they will be replaced by normal cells, and the process starts all over.

Q. Thank you, Doctor.

Are we threw with this one, Doctor?

(Pause.)

BY MR. SHEFFLER:

Q. Doctor, I believe the third line of evidence that we have not yet talked about that supports your opinion that Mr. Kotler would have avoided his cancer had he quit smoking in 1966 was the statistical evidence.

Would you briefly tell us how that supports your opinion?

A. Yes. I think that there are numerous published studies that show when a smoker quits smoking his risk of developing lung cancer decreases over time, and eventually approaches that of a non-smoker.

Q. Do the studies indicate a time period where this would normally take place?

A. Yes, they did.

Q. How long would it take for a decline in risk to approach that of a non-smoker?

A. It varies according to the study that you read. Of course everybody has their own opinion, but I think a generally accepted time period to

approach baseline is approximately ten to fifteen years.

Q. Doctor, based upon your review of the medical records and medical materials and your knowledge of cell biology, molecular biology and statistical evidence, when do you believe Mr. Kotler's cancer began?

A. Well, that's a tough question, because we have to break it up into two parts.

If I may stand up.

We have to realize that from the time his first cancer cell became -- his first cell that became cancerous, and then had a -- not a latency period, but a clinical period, but it multiplied and became a clinical mass, that's one stage, and based upon my review of the records and his various x-ray data, this probably took four to five years from the first cancer cell dividing, doubling, becoming large enough to becoming clinically cancer. That's approximately four to five years.

Then we have to also say, well, when did the genetic events to push this lung cell into cancer occur, and that probably takes approximately five to seven years for a cell to accumulate enough

errors to become cancer.

Q. That five to seven year period, Doctor, what would happen in the five years if the cell dividing were removed?

A. If they were removed and he was lucky enough not to have any more, then the cell would either die out or be repaired, and it would not go on to cancer. Once it gets cancer, it divides, and you'll see the clinically evident mass in four to five years.

Q. Were there cells in Mr. Kotler's lungs in 1966 that had genetic alterations that would lead to his cancer in 1984?

MS. LUMSDEN: Objection, unless he has an opinion to a reasonable degree --

THE COURT: Excuse me?

MS. LUMSDEN: I would object unless he has an opinion to a reasonable degree of medical certainty.

THE COURT: Yes.

BY MR. SHEFFLER:

Q. Doctor, do you have an opinion to a reasonable degree of medical certainty whether or not there were cells in Mr. Kotler's lungs in 1966

that had any role with his 1984 cancer?

A. Yes, I have an opinion.

Q. Would you please tell the jury what that opinion is?

A. I believe that he had cells that were genetically damaged in 1966, but these cells were not the same cells that eventually went on to give him his clinically evident cancer.

The cells in 1966 would have long either washed out, died out or been repaired.

Q. Doctor, there has been some discussion in this case about metaplasia and dysplasia.

Are you familiar with those concepts?

A. Yes, I am.

Q. Could you explain those for the jury?

A. Yes. I'd like to bring back one of my photos.

Q. Sure.

(Pause.)

A. Metaplasia and dysplasia are terms that we've reserved for epithelial life, and in the lung we usually associate them with the epithelial line in the air tubes.

By definition metaplasia is one mature

cell type changing into another mature cell type. In the case of the epithelium of the lung, it is usually the tall columnar cells of the bronchi being irritated, flattening out and assuming that of a squamous, a mature squamous cell. That is metaplasia.

Dysplasia, on the other hand, is an abnormal maturation of these multiple layers of cells, so when the precurser stem cell divides it doesn't really give rise to a mature cell, it's a more immature cell, and you get a whole population that are less mature than normal. And we can actually see this process. It looks a little altered under the microscope.

Q. Doctor, this dysplasia occurs in the air tube, is that correct?

A. Yes.

Q. Does that have any relationship to any specific type of cancer?

A. It's usually associated with squamous cell carcinoma.

Q. Doctor, in the periphery, could you show us where the periphery is again?

A. Again, the periphery is out here in the

alveolar air sacks.

Q. Does dysplasia occur in those air sacks, Doctor?

A. Many people have looked for dysplasia equivalents in the periphery of the lung, and it's never been found.

Q. Doctor, what is the type of cancer most frequently found in the periphery of the lung, in your opinion?

A. Well, we know from numbers by looking at lung cancer cases in the periphery it's usually large cell carcinoma or adenocarcinoma.

Q. Doctor, in your opinion, where did Mr. Kotler's cancer arise?

A. It arose out in the periphery.

Q. And what type of cancer did Mr. Kotler have?

A. He either had a large cell carcinoma or a poorly differentiated adenocarcinoma.

Q. What's your basis for saying Mr. Kotler's cancer arose in the periphery?

A. We have several lines of evidence. We have, of course, the x-ray evidence, but we also have the surgeon's report, that we actually stuck a

tube down the air tubes and looked around and they all looked clean as far as he could tell. So he certainly didn't have a central squamous cell carcinoma.

Q. Doctor, what does metaplasia and dysplasia have to do with Mr. Kotler's cancer?

A. In this instance nothing. He did not have a classic squamous cell carcinoma, which is associated with metaplasia and dysplasia. He had peripheral cancer.

Q. Thank you very much, Doctor.

MR. SHEFFLER: No further questions.

CROSS EXAMINATION

BY MS. LUMSDEN:

Q. Doctor McHugh, this fellowship that you did at the Roche Institute in Nutley, New Jersey, does that have any relation with Hoffman LaRoche?

A. Yes, it does.

Q. Hoffman LaRoche is an international drug company, is it not?

A. Yes, it is.

Q. At least its American base is in Nutley, New Jersey?

A. Yes, it is.

Q. And the research that you did at that time had to do with the effects of retinoids on tumor stem cells?

A. Retinoids with vitamin A.

Q. And is retinoid, is that a synthetic form of vitamin A?

A. No. Actually this is the vitamin A. It's concentrated, but this is the stuff that you can buy in a health food store.

Q. Now, Hoffman LaRoche produces a drug that is a synthetic vitamin A, do they not?

A. They produce a whole series of retinoid compounds, yes, they do.

Q. And one of them is the drug called Acutane?

A. Yes, it is.

Q. And that's a synthetic vitamin A drug, is it not?

A. It's thirteen CIS, C-i-s, CIS being the chemical configuration of the chain, whether it's sitting like a chair or whether it's flat.

Q. Now, the effects of vitamin A on the stem cells that you studied, were those cancerous effects?

A. Those were cancerous stem cells, yes.

Q. Okay. But you were studying -- in other words, does vitamin A -- are you saying that vitamin A causes carcinoma?

A. No.

Q. In other words, that it's an agent that causes carcinoma?

A. No, not at all. In fact, you can treat certain cancers with vitamin A. I think that there is a recommendation to eat a lot of yellow vegetables to decrease your risk of cancer, at least in the nutritional literature, so this is a beneficial agent.

Q. But vitamin A can in large doses cause other types of tumors and problems, can it not?

A. It cannot cause tumors, but it can be toxic.

Q. How about teratogenesity?

A. Yes.

Q. What is that, Doctor?

A. Chemical effects on the developing embryo. In fact, if I may, we have -- my March of Dimes grant that address this issue very specifically, because of the profound effects of vitamin A on the early embryonic cell it can cause premature

differentiation of the cell and actually arrest embryonic development.

Q. In fact, the drug Acutane that Hoffman LaRoche sells is known to cause such birth defects, does it not?

A. Yes, it does.

Q. Does your research have anything to do with the Hoffman LaRoche Drug Company?

A. No, it doesn't.

Q. They are affiliated with the Roche Institute of Molecular Biology, are they not?

A. The history of the Roche Institute was that it was started by Doctor V.D. Matte who late in the sixties took a lot of the profits produced by Hoffman LaRoche and set up a research institute off the main campus devoted to molecular biology research.

Q. Now, the cellular differentiation changes that your area of research is in is in genito urinary tumors, is that correct?

A. The cells that I use are what are known as embryonal carcinoma cells.

Q. And are those bronchial cells related to the lungs?

A. These are very, very interesting cells. If you take a single embryonal carcinoma cell --

THE COURT: How about answering the question.

Are they bronchial or not?

THE WITNESS: They are related.

BY MS. LUMSDEN:

Q. Doctor, you have written about retinoids and retino acid as well, is that right?

A. Yes.

Q. That's a particular area of interest of yours?

A. Yes, it is.

Q. Now, if I may, you have -- strike that.

Have you, yourself, written about the pre-cancerous changes on stem cells, on bronchial stem cells?

A. No, I haven't.

Q. Are you familiar with any of the literature in that area?

A. Somewhat.

Q. Does the name Doctor Auerbach -- is that name familiar to you?

A. Not real familiar.

Q. But you're aware that Doctor Auerbach has written on that very subject?

A. If you gave me some specifics.

Q. Well, Doctor Auerbach and others have done research, have they not, on the changes in the cells, in the bronchial cells of smokers, are you aware of that?

A. I'd have to see the paper.

Q. (Handing).

(Pause.)

A. Yes. These are the cells of the bronchi.

BY MS. LUMSDEN:

Q. And Doctor Auerbach in this particular study made some determinations as to the number of years that, where you have these arrows, to the number of years that go between atypia, mild atypia, moderate atypia, and marked atypia, and then on to carcinoma in situ in smokers, did he not?

A. Again, he is dealing with the cells of the bronchial epithelium, and specifically addressing squamous cell carcinomas.

Q. But these studies have to do with bronchial carcinomas, do they not?

A. With carcinomas arising from the air tube

epithelium.

Q. Okay. And you're saying the basis of your opinion is that it arose in the air sacks?

A. No, no. I'm not saying that at all. I just said that if one removes the insults from the epithelium, if you no longer can expose the epithelium, then they are reversible.

Q. Now, would you agree that there are three degrees of atypia, namely mild, moderate, and severe, or marked?

A. In the bronchial epithelium, yes.

Q. And the marked atypia, is that what some observers refer to as dysplasia?

A. Atypia is a term that we use to describe individual cells. Dysplasia is a term that we use to describe epithelial organs.

Q. Well, in this study -- strike that.

I'm going to ask you if you agree with the following conclusion of Doctor Auerbach. "While following subjects with marked atypia, it has been observed that very few, if any of them, return to milder atypia without subsequently reverting to marked. This has lead us to postulate that severe atypia may represent an irreversible step in the

progression to carcinoma."

MR. SHEFFLER: Objection, your Honor.

THE COURT: I'll hear you.

(Side bar conference.)

MR. SHEFFLER: My objection, your Honor, is foundation for reading the article to the witness since there's no -- there has been no record that he's seen, relied upon it or used that. She showed it to him now.

THE COURT: We don't have any testimony that this fellow is a recognized expert.

MS. LUMSDEN: He's familiar with the literature in the area, your Honor.

THE COURT: I'm sure there's a lot of literature in the area, some of which is junk. You would have to establish that this isn't the junk.

MR. SHEFFLER: When was the article written?

MS. LUMSDEN: Nineteen seventy-four.

MR. SHEFFLER: Well, I don't know if there's any record. When the article was written might have something to do with his opinion as well, your Honor.

MS. LUMSDEN: This Doctor, number

one, doesn't specialize in pulmonary pathology.

THE COURT: That's fine.

MS. LUMSDEN: Doctor Mark said this is the guy who's the authority.

MR. LANE: On squamous.

THE COURT: Why don't you ask him the question?

MS. LUMSDEN: He has given an opinion, and I'm trying to get at the basis.

THE COURT: Why don't you ask him the question if this is a --

MS. LUMSDEN: He's going to say he doesn't know.

I still believe that I have a right, your Honor, if he's talking about being familiar with the literature in the area, to bring up --

THE COURT: I don't think you can come in with an article from Popular Science or the New York Times Sunday Magazine.

MS. LUMSDEN: This is from Cancer.

I'll lay some foundation.

THE COURT: Lay some foundation.

(End of side bar discussion.)

BY MS. LUMSDEN:

Q. Doctor McHugh, is it fair to say that you recognize Doctor Auerbach as someone who has written in this area of carcinoma of the lung?

A. This specific paper you showed me was squamous cell carcinoma, yes.

THE COURT: Do you recognize this author as a reliable authority in the field on which he's writing?

THE WITNESS: Yes.

THE COURT: Okay.

BY MS. LUMSDEN:

Q. Now, in this study of Doctor Auerbach, he computed the mean years that mild atypia lasted, did he not?

MR. SHEFFLER: Objection, your Honor. Objection.

THE COURT: Objection is sustained. I believe the witness said he hasn't read the article..

MS. LUMSDEN: Can I show him the article, your Honor?

THE COURT: Sure.

BY MS. LUMSDEN:

Q. (Handing).

A. Did these people quit smoking?

Q. Let me give a foundation first just to state what the study is about.

This study, Doctor, included men who had ever smoked cigarettes, cigars or pipes on a regular basis were considered smokers. This term included former smokers.

A. We don't know whether -- I think I saw uranium miners in there.

Q. Those were another category. They also studied uranium miners, then they studied non-miners, all of which had some smoking history.

A. Do we know that they were removed from the environmental influences?

Q. Yes.

Q. Men who had ever worked in a uranium mine were considered uranium miners, all others were non-miners?

A. We don't know whether the population he studied had ceased all smoking.

Q. No. Some were smokers and some were former smokers.

A. Okay. Some are current smokers and some were former smokers.

Q. Now, Doctor, I'd just like to have you look at this chart relating to the non-miner smokers. Now, this study began in 1957, they started collecting data from the sputum of these subjects every three months, and the study lasted from 1957 until the time of publication.

Now --

MR. SHEFFLER: Objection, your Honor. I didn't hear a question.

THE COURT: I didn't hear any question either.

MS. LUMSDEN: I'm sorry, your Honor.

BY MS. LUMSDEN:

Q. Cytologic examinations of sputum collected periodically since 1957. Okay. Now --

THE COURT: Okay what?

MS. LUMSDEN: I'm giving him the background for the next question, your Honor, in terms of how long they collected these sputum samples.

MR. SHEFFLER: Objection, your Honor.

THE COURT: Sustained.

BY MS. LUMSDEN:

Q. As far as the --

THE COURT: The objection is sustained.

Have you got something there that you want to read to him and ask him if he agrees with it?

MS. LUMSDEN: That's what I'm doing right now, your Honor.

As far as the --

THE COURT: Long way around. Isn't there a shorter route to where you want to go?

BY MS. LUMSDEN:

Q. Well, the list here of the mean years of mild atypia is listed as three years.

Would you disagree with that in terms of the lengths of time that mild atypia would last?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Overruled.

THE WITNESS: I'm sorry?

THE COURT: The objection is overruled. If you understand the question and can answer it, answer it.

A. What I understand from this paper is that he has got a population that he has not segregated out as to whether they have continued smoking or not.

But what I gather from that paper that you showed me is that it's taken three years of some type of environmental risk to give them atypia, and I agree with that, sure.

BY MS. LUMSDEN:

Q. Then from mild atypia to moderate atypia, he has a mean range of 3.9 years.

Do you agree with that?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Overruled.

BY MS. LUMSDEN:

Q. As to whether or not that's a reasonable period of time from mild atypia to moderate atypia?

A. I think in the model that I have proposed, that these people are picking up the genetic hits on a continuing basis and they are getting their atypia, sure, their dysplasias.

Q. And then would you agree that their finding of a mean year of marked atypia from that another 1.2 years, would you agree with that being within the normal range?

A. Yes.

Q. And then mean years to carcinoma in situ they have listed as one year.

Is that a reasonable period of time, in your opinion, to go from the marked atypia to the carcinoma in situ?

A. The total number of years?

Q. The mean years from mild atypia to carcinoma in situ are listed as 9.2 years?

A. I think if you keep on smoking and have the exposure, yes, it's reasonable to say that you can go from a normal cell to an abnormal cell in that time.

Q. Now, would you agree with this conclusion of the study, that "cessation of either cigarette smoking or mining has no effect on the progression of atypias to carcinoma in situ"?

A. Well, again since he hasn't really controlled for that, I don't know how he can make that statement.

Q. He has controlled in that there is what he has termed a smoking index. In other words, when, age is starting to smoke, how long smoked.

MR. SHEFFLER: Objection, your Honor.

May we see you at side bar?

MS. LUMSDEN: I'll withdraw that

question, your Honor.

BY MS. LUMSDEN:

Q. Would you agree, Doctor, that tobacco smokers have much lower prevalence of normal cytology and much higher prevalence for both moderate and marked atypia than do non-smokers?

A. Again, we have to know what they mean by the term "atypia." As a person who does cytology, I believe that's about a 1973 paper, I think that I had just graduated from college then.

Atypia, we have inflammatory atypia that he has signs of. If he says dysplasia, that's something different to me.

Q. He doesn't use the term dysplasia.

As mild atypia -- well, he starts out with the definition of regular metaplasia, cells all of about the same size, and goes on, up to marked atypia, which he defines as "cells vary markedly in size, but are generally larger than those in moderate atypia. Nuclear pleomorphism is marked; nuclear material is coarse and sometimes clustered around about nuclear membrane. N/C ratio varies markedly." Namely the nuclear to the cytoplasm ratio. "It may be higher or lower than normal.

Nucleoli are present, but are small and may be acidophilic. Acidophilic cytoplasm predominates. Single cells predominate."

Do you agree with that as a reasonable definition of marked atypia?

A. Again, I don't know the cytologic standards back in 1973.

I do know today that we break up dysplasia from atypia on a routine basis. Atypia carries no flags for the clinicians that I give my report to. If I say dysplasia, then the light goes off and then they are very worried about that. I'm not -- I just don't know back in '73 their terminology.

Q. This paper, just for the record, was in '74, but I doubt if that makes a difference in your opinion, is that right, that you've just given?

Doctor, are you familiar with, I can show this to you, another study by Doctor Auerbach from the New England Journal of Medicine, July 19th, 1962, entitled Changes In Bronchial Epithelium In Relation To Sex, Age, Residence, Smoking & Pneumonia?

MR. SHEFFLER: Objection, your Honor.

THE COURT: What's the question?

MS. LUMSDEN: Just if he's familiar with the study, your Honor. I'm just showing it to him.

A. No, I'm not.

BY MS. LUMSDEN:

Q. But Doctor Auerbach you recognize as an authority who's written in this area, is that right?

A. True.

Q. Would you agree with Doctor Auerbach's findings of that "we found a high degree of relation between cigarette smoking and certain changes in the bronchial epithelium of men who have died of causes other than lung cancer"?

Do you agree that that happens in smokers?

A. Yes.

Q. And "these changes included hyperplasia, loss of cilia, metaplasia and the occurrence of cells with atypical nuclei"?

A. Yes, that seems reasonable.

Q. "What impressed us most was the finding of many lesions composed entirely of cells with atypical nuclei in the bronchial epithelium of cigarette smokers, the number of such lesions increasing with amount of smoking."

Do you agree with that, Doctor?

A. That they were impressed by that?

Q. No.

Do you agree that that finding is within what your -- reasonable with what you know about changes in the epithelium of the bronchus in smokers?

A. If you define the changes by the microscope, again back in '62, I think that's very reasonable.

Q. Doctor, I'd like to ask you if you've seen -- if you're familiar with this paper by Doctor Auerbach published in the New England Journal of Medicine on February 22nd, 1979 entitled Changes In Bronchial Epithelium In Relation To Cigarette Smoking, 1955 to 1960 versus 1970 to 1977 (handing).

(Pause.)

A. I might have, but I'm not intimately familiar with this. I was reading the New England Journal at this time.

BY MS. LUMSDEN:

Q. I'm going to ask you if you agree with this conclusion by Doctor Auerbach.

"Basal cell hyperplasia, a reversible change, is presumably a reaction to some deleterious factor; it is probably protective rather than harmful. Loss of cilia, a reversible change, presumably results from the presence of some deleterious factor; it is harmful in that it destroys one of the mechanisms by which foreign material is ordinarily removed from the lungs.

"In our opinion, the occurrence of atypical nuclei is the first definite step along a road that may eventually lead to carcinoma in situ and from there to invasive carcinoma."

Do you agree with that, Doctor?

A. Yes.

Q. Now, Doctor, when you talk about the reversibility of cell changes, are you saying that a cell with marked atypia or with an atypical nucleus is going to change back to its normal cell, as it started?

A. If a cell that has been injured is removed from all other injurious agents, it will revert back to normal.

Q. So you're not talking about these cells dying off or being fought off by the body's immune

system and replaced with normal cells, you're talking about a cell actually changing back?

A. Well, there's two things that are going on, depending upon which cell population is effected and what the changes are.

Q. So some of those changes are not reversible in that the cell actually doesn't go back to the way it was, but it's the body's immune system that may fight off some of those cells and replace the cell population with normal cells?

A. We've got three things going on in that question, and I'd like to avoid any type of immune -- immunologic reaction at all in my discussion.

Two things are going on. Either the cell that has been genetically altered is going to repair itself, or it's going to go ahead and die out in its normal life course.

Q. Now, in your opinion, are there differences between individuals as far as how long these changes take place and whether or not some changes become irreversible or not?

A. I think there's always biological differences in individuals.

Q. Now, you talked a moment ago on direct

examination that in your opinion it was five to seven years for the cell to become -- to have errors enough to become cancer, is that correct, five to seven years?

A. If you're unlucky.

Q. And is that comparable to the nine year period that Doctor Auerbach was referring to; in other words, up to the point of carcinoma in situ?

A. I don't know that.

Q. Now, you then said in your opinion it would take another four to five years from the first cancer cell to becoming clinically evident, is that correct?

A. Yes.

Q. And clinically evident, Doctor, would be a lesion of the size of approximately one centimeter?

A. Yes.

Q. And when you talk about multiplication to that point, and your estimate of four to five years, are you talking about the doubling time of that tumor?

A. Yes.

Q. And do you recognize Doctor William Weiss to be an authority in the field of doubling times on

tumor growth rates?

A. He has written in the environmental journal on this, yes.

Q. Do you recognize him as a reliable authority?

A. Yes.

Q. Now, according to -- are you familiar with his article that appeared in the Journal of Occupational Medicine in May of 1984 entitled Implications Of Tumor Growth Rate For The Natural History Of Lung Cancer?

A. May I see the article?

Q. (Handing).

(Pause.)

A. Yes, I'm familiar with this paper.

BY MS. LUMSDEN:

Q. Now, Doctor Weiss in this paper gives a doubling time for large cell carcinoma of 86 days, does he not?

A. If you say so.

Q. Well, does that sound within the realm of what's known about doubling times for large cell carcinomas?

A. Yes. Depending upon the individual tumor.

Tumors can double anywhere from eight days all the way up to 86 days.

Q. And beyond 86 days?

A. Unlikely.

Q. Unlikely for any lung tumor?

A. Unlikely for any cancer. Whether we say 86 or 90 days, that's fairly long.

Q. Well, in this particular paper the doubling time for adenocarcinoma was found to be 161 days.

Do you agree that that's within the range of what's known for adenocarcinomas?

A. Again, every tumor has its own individual characteristic, and what one has to do is look at that tumor individually. Again, there are ranges.

Q. And these ranges for an undifferentiated carcinoma of the lung would be anywhere from 33 to 480 days, would they not?

A. Yes.

Q. So if we take a mean, Doctor, as Doctor Weiss did, of 86 days and we -- am I correct that to reach the size of a one centimeter tumor which would be clinically visible would take 30 doublings, approximately?

A. Approximately, yes.

Q. And if you multiply -- if you multiply that out, doesn't that then in the case of a large cell carcinoma, if you're using a doubling time of 86 days, indicate that to go from one cell to one centimeter takes in excess of seven years?

A. If you're talking about tumors in general and using a mean from all the reported literature.

However, I think one would basically be on firmer ground if you look at the data in the individual case. I think that you could probably do similar calculations if you would look at the medical records from Mr. Kotler. I think that you have data points there by looking at the chest x-ray to see that he has clear x-ray in '81, possibly a clear x-ray in early '84, and then he starts to get a mass. Whether the mass is the size of a marble sometime in mid '84 to grow to the size of an orange in late of '84, I think that -- and I have not done this, but I think if you would do this you could probably come up with a straight line with a proper slope and figure and calculate back and figure out that his doubling time is probably in the order of 40 days.

But I agree with what you're saying in

general, if you picked 80 days for any large cell carcinoma, but I'm not sure how that applies since we have hard data in this case.

Q. Well, Doctor, you say he had a clear x-ray in 1981?

A. The record that I read said that he did not have a tumor in '81.

Q. Did he have a clear chest x-ray in 1981?

A. He did not -- I don't know that. I know that he did not have a tumor, according to the x-ray report.

Q. You saw an x-ray report from 1981 on George Kotler?

A. Yes.

Q. From what hospital?

A. I don't recall.

Q. And you say that he had a clear chest x-ray in early 1984?

A. The x-ray report that I read said that, yes. I think that there has been testimony that in retrospect there probably was a tumor.

Q. You looked at those films, didn't you?

A. Yes. I'm not an expert radiologist, but I read the reports.

Q. Did you read the testimony of Doctor Hilgenberg who said it was virtually the same size in May of 1984 as it was in October of 1984?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Sustained.

BY MS. LUMSDEN:

Q. Were you shown the trial testimony of Doctor Hilgenberg?

A. Yes.

Q. And do you recall Doctor Hilgenberg testifying that the tumor was virtually the same size in May of 1984 as it was in October of 1984?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Do you quarrel with the --

MR. SHEFFLER: I quarrel with the characterization of the testimony.

THE COURT: Read it right from the transcript. Do you have the transcript?

MS. LUMSDEN: Yes, I do, your Honor.

THE COURT: Read the transcript, and we won't have any problem.

BY MS. LUMSDEN:

Q. Do you recall reading his statement that it

was a four month interval and it looked like there was very little change in its appearance on the chest x-ray? Do you recall that testimony?

A. No, not specifically.

Q. Now, you yourself, Doctor, have taken mean ranges and doubling times and applied them to a particular case, have you not?

A. I have given you the basic data that we know from DNA repair from experimental studies in the laboratory.

Q. I'm talking about mean doubling times.

Haven't you taken data specifically from this article by Doctor Weiss and applied it to a particular case?

A. No.

Q. Didn't you testify on behalf of the American Tobacco Company in another trial?

A. Yes, I've testified once.

Q. And in that trial, didn't you, in fact, cite the data from Doctor William Weiss and cite the mean doubling times in this article and apply them to Mr. Gonzales' tumor in that case?

A. We applied his method.

Q. And you had information about Mr. Gonzales'

tumor just as we have information about Mr. Kotler's tumor, did you not?

A. We had information about his x-rays, yes.

Q. And if you take the mean doubling time of 86 days and compute it out, will you agree that by the time you reach the clinical stage, one centimeter, it would be in excess of seven years?

MR. SHEFFLER: Objection, your Honor.

We've already gone through this and he's already explained why it's not appropriate.

THE COURT: Well, he can do it.

Do you have an answer to that question?

THE WITNESS: My answer is that we have -- or I have used actual data points based upon the clinical course of the patient.

BY MS. LUMSDEN:

Q. Now, Doctor, from one centimeter to reach another four centimeters would take another six doublings, would it not?

A. Yes.

Q. And Mr. Kotler's tumor when it was detected was four by seven centimeters, is that right?

A. I don't have the numbers at my fingertips.

Q. I'll ask you to assume that according to the CAT scan report that's in the hospital record the dimension of that tumor was four by seven centimeters.

Now, to reach just the four centimeter diameter --

A. Four or seven?

Q. Four by seven.

A. The greatest diameter is?

Q. Seven.

A. Seven.

Q. So to go from the one centimeter clinically detectable size up to just a four centimeter size would take in excess of two years, if you calculated based on the meantime of 86 doublings, is that right?

A. I've looked at it differently. I went from one centimeter to seven centimeters rather than one to four.

Q. All right. And if you go from one to seven and you use the mean doubling time of -- how many doubling times would it take to go from one centimeter to seven centimeters?

A. You told me six.

Q. That was to four, is that correct, to four centimeters, from a tumor of a diameter of one centimeter to a tumor of a diameter of four centimeters, it takes six doubles, am I right?

A. Okay.

Q. Am I right about that?

A. I'd have to sit down with a pencil and paper and you have to work out four square pie I cubed, volume of the square.

Q. Let me ask you if you agree with this.

The relationship between the diameter and volume of the sphere is such that the volume doubles three times as the diameter doubles once, thus a tumor approximating a spherical mass with a diameter of one centimeter will increase its diameter to 1.26 centimeters in one doubling of volume, 1.59 in two, two centimeters in three, 2.52 centimeters in four, 3.18 centimeters in five and four centimeters in six.

Do you agree with that, Doctor?

A. Yes.

Q. All right. So it takes six doublings to get up to the size of four centimeters, is that correct?

A. Sure.

Q. From one centimeter?

A. Yes.

Q. And that takes, on top of the seven plus years we already have another two plus years to get to the four centimeters, is that right?

A. Well, let me back up and say he went from clinically undetectable -- you say one centimeter to four by seven centimeters in how many months?

Q. Well, the clinically undetectable stage, the only x-ray that has been -- report that has come into this case for 1976 --

MR. SHEFFLER: Objection, your Honor.

May we see you at side bar on this?

THE COURT: Yes.

(Side bar conference.)

MR. SHEFFLER: Your Honor, the representation, I believe, is inaccurate in that the records from Cambridge Hospital have a 1981 report of chest film.

THE COURT: What did counsel say?

MR. SHEFFLER: She said the first one was '76.

MS. LUMSDEN: Well, first of all, these records aren't in evidence.

THE COURT: You can't make representations if something isn't in the record.

MS. LUMSDEN: No. In evidence in this case, your Honor.

THE COURT: In the record. Is there a record of a '76?

MR. SHEFFLER: The '76 isn't in the record either then.

THE COURT: Is there a record, an x-ray in 1976?

MR. SHEFFLER: My objection is that it was 1981, your Honor.

MS. LUMSDEN: I'll ask him if he reviewed a film from '76, your Honor.

THE COURT: Were there any films in '76?

MS. LUMSDEN: Yes.

MR. SHEFFLER: I know there was one in '81. I don't have any record of '76.

THE COURT: What evidence do you have that there was a film in '76?

MS. LUMSDEN: I'll withdraw it, your

Honor.

THE COURT: Do you have it or don't you have it?

MR. INGE: Seventy-five was the film.

MS. LUMSDEN: Seventy-five. That's the Doctor Dow film, your Honor. There has been testimony about that.

MR. SHEFFLER: Doctor Dow's films do not exist. We tried to get the films, he destroyed them. There is a report.

THE COURT: You can make a reference to the report.

Let me ask you whether you think there's anything really valuable that you're getting out of all of this. This is terribly bogged down in centimeters.

MS. LUMSDEN: Your Honor, the point is how long his tumor was present. In other words, we have --

THE COURT: Why is that a point?

MR. SHEFFLER: He said five and they are saying seven. What difference does it make?

THE COURT: I have some question --

MS. LUMSDEN: We're not saying

seven. We're saying more like nine or ten years, your Honor.

THE COURT: What's the significance of all this?

MS. LUMSDEN: It brings you back to 1974, and then you have the length of what Doctor Compton and Doctor Auerbach has testified are irreversible changes.

THE COURT: There's some problem with this stuff, because you're making computations. It sounds to me, at least, as if they are based upon a spherical tumor, and what we have here is a kind of a -- not a grapefruit size, more or less lemon shaped, I suppose, or egg shaped with an eliptical profile. I don't know what that does to all these calculations, but I suspect it does something, probably in your favor. If you want to work it out.

I think there's some possibility of confusion. I don't know whether it's worth straightening it out or not.

MR. SHEFFLER: Your Honor, the problem I have with reading and trying to use this witness to compute her testimony from these articles

that he's already said he didn't use the mean, he had data points, and now she's trying to have him compute out these figures.

MS. LUMSDEN: He used the mean. In the Gonzales case he used the mean.

THE COURT: I'm not stopping you from doing all this. I myself, if I were sitting as a finder of fact, would at this point be driving very little benefit from it. If you think it's important, but try not to get bogged down if you can help it. I'll let you go ahead with it. You've got to get your postures right.

MS. LUMSDEN: I think I've gotten out the points I was trying to make.

THE COURT: Get your posture right when the x-rays were taken you can go ahead.

MR. SHEFFLER: Thank you, your Honor.

(End of side bar discussion.)

BY MS. LUMSDEN:

Q. Doctor, if you take the data from the Auerbach article that I showed you, would you agree that there is data to support a -- from accepted authorities to support the length of time from mild atypia to carcinoma in situ as being in excess of

nine years?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Overruled.

A. I think with continual environmental exposure, that's correct.

BY MS. LUMSDEN:

Q. All right. And you disagree, then, with the conclusions of Doctor Auerbach that the cessation of either cigarette smoking or mining has no affect on the progression of atypias to carcinoma in situ?

A. I think that the current --

Q. Do you disagree with that?

A. Yes.

Q. Now, will you also agree, Doctor, that based on mean doubling times, in using the mean of Doctor Weiss which you yourself used in your prior testimony, that this tumor from that point after carcinoma in situ was some period of time, then changed into its first invasive tumor cell, that tumor then, according to the calculations of Doctor Weiss, was in existence in excess of nine years by the time it was then detected in 1984?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Overruled.

A. No, I disagree with that. We did not use those numbers. We used his method. The numbers were gotten from the clinical records.

BY MS. LUMSDEN:

Q. Didn't you in that case use the doubling time of 30 days?

A. If I can draw on the board I can show you how that was done.

Q. Could you answer my question?

Did you in that case use a doubling time of 30 days?

A. Yes.

Q. And you applied it to Doctor Weiss' formula, did you not?

A. We applied it to his formula.

Q. By the way, did you get paid by the American Tobacco Company for your testimony in that case?

A. I got paid by Chadbourne & Parke for my time spent preparing. Nobody paid me to say anything. My testimony is my own. They paid for my time.

Q. Did you get paid for your time by the

American Tobacco Company, Chadbourne & Parke in that case?

A. Yes, I did.

Q. How much did you get paid?

A. My fee was $200 an hour.

Q. What was your total charge?

A. I don't recall.

Q. Can you give us your best estimate?

A. I think it was $6,000 perhaps.

Q. How many hours did you put into that case, Doctor?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Just asking him to do arithmetic, I suppose.

A. Thirty hours is the arithmetic.

BY MS. LUMSDEN:

Q. Are you charging the same hourly rate in this case?

A. Yes, I am.

Q. And how many hours have you put into this case so far?

A. Approximately twenty.

Q. When were you first consulted on this case?

A. Many months ago.

Q. Was it after the Gonzales trial?

A. Yes, it was.

Q. Doctor, is it your testimony that the cancer in this case arose in the tiny air sacks?

MR. SHEFFLER: Objection. Asked and answered.

THE COURT: Overruled.

Is that your testimony?

THE WITNESS: It's my testimony that he had a peripheral cancer.

BY MS. LUMSDEN:

Q. And large cell carcinomas are known to appear in the periphery of the lung, are they not?

A. Yes, they are.

Q. And there are bronchi considered in the periphery of the lung, are there not?

A. There are terminal bronchioles that branch out, and bronchi have generations of branching, like branching on the tree.

Q. Did you read or do you recall reading in Doctor Hilgenberg's trial testimony as to what he meant by the peripheral location of the tumor?

A. I don't recall.

Q. Do you recall, see if this refreshes your

recollection, him saying that the man's tumor -- "so again I think of this man's tumor as being peripheral, because it was beyond or out in the lung, beyond where these main bronchial tubes are located, beyond where I could see with this bronchoscope"?

A. I agree with that.

Q. There are other divisions of the bronchi, are there not, in that area, that would have bronchial epithelium?

A. The bronchial epithelium thins out as it goes down the tree, and it becomes, say, from six to eight layers all the way down to one or two.

Q. But there is bronchial epithelium?

A. It's transitional epithelium more accurately.

Q. But it is epithelium?

A. Yes, epithelium.

Q. Doctor, is lung pathology something that you particularly specialize in?

A. I don't subspecialize in lung pathology.

MS. LUMSDEN: Thank you.

No further questions.

THE COURT: Redirect?

MR. SHEFFLER: I have just a couple, your Honor.

THE COURT: Sure.

REDIRECT EXAMINATION

BY MR. SHEFFLER:

Q. Doctor, the articles that Miss Lumsden showed you from '74 and '62 and the other, I think it was '79, what type of cancers were those?

A. Those were squamous cell carcinomas.

Q. And what type of cancer did Mr. Kotler have?

A. He had a large cell carcinoma or a poorly differentiated adeno.

Q. The articles again by Doctor Auerbach that Miss Lumsden was showing you, where were those cancers arising?

A. Arising in the bronchi, because they were getting bronchial cells from cytology.

Q. And the bronchi is again, Doctor, is that the air tubes?

A. That's the center portion of the lung.

Q. And where was Mr. Kotler's cancer?

A. It was out beyond where they could get the cytology of the bronchi. It was out in the

periphery.

Q. Doctor, Miss Lumsden asked you whether there was periphery -- excuse me.

Miss Lumsden asked you whether there was epithelium in the periphery.

Is there epithelium in the periphery, Doctor?

A. It's comprised of the type one and type two pneumocytes.

Q. Is the epithelium made up of different cells in the periphery than the epithelium in the air tubes?

MS. LUMSDEN: Objection. Leading.

THE COURT: Overruled.

THE WITNESS: I'm sorry?

THE COURT: The objection is overruled. You can answer the question.

A. Yes. As you can see from the picture, the type one and type two pneumocytes look different and act different than the epithelium of the air tubes.

MR. SHEFFLER: No further questions.

THE COURT: Recross?

RECROSS EXAMINATION

BY MS. LUMSDEN:

Q. Doctor, if you take Doctor Hilgenberg's definition of what he meant by peripheral, meaning beyond the main bronchus, there are bronchi outside in the periphery that have epithelium in there, do they not?

A. Yes.

Q. And the tumor in this case was in the center of the chest and it involved the mediastinum, did it not?

MR. SHEFFLER: Objection, your Honor.

THE COURT: Overruled.

A. According to the records it spread into the mediastinum.

BY MS. LUMSDEN:

Q. Thank you.

THE COURT: Is that it? Anything further?

MS. LUMSDEN: No.

THE COURT: Thank you, Doctor.

That will bring us about to the time for the morning recess, which we will now take.

(Jury out of courtroom at 10:50 o'clock